### WEST

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DB=USPT,PGPB; PLUR=YES; OP=AND				
<u>L7</u>	15 and L6	16	<u>L7</u>	
<u>L6</u>	infus\$ near6 artery	1296	<u>L6</u>	
<u>L5</u>	13 and L4	675	<u>L5</u>	
<u>L4</u>	(coronary or sinus) adj (artery or blood adj vessel)	15567	<u>L4</u>	
<u>L3</u>	11 and L2	1747	<u>L3</u>	
<u>L2</u>	(cardiovascular or cardiac or heart) near3 (disease or disorder or defect)	28230	<u>L2</u>	
<u>L1</u>	adeno-associated adj virus or aav	6380	<u>L1</u>	

**END OF SEARCH HISTORY** 

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## Search Results - Record(s) 1 through 16 of 16 returned.

1. 20030148968. 03 May 01. 07 Aug 03. Techniques and compositions for treating cardiovascular disease by in vivo gene delivery. Hammond, H. Kirk, et al. 514/44; 604/500 A61K048/00 A61M031/00.
2. 20030147862 . 16 Oct 00. 07 Aug 03. Methods for the modulation of neovascularization and/or the growth of collateral arteries and/or other arteries from preexisting arteriolar connections. Buschmann, Ivo R., et al. 424/93.21; 514/12 514/44 A61K048/00 A61K038/18.
☐ 3. 20030100889. 03 Jul 02. 29 May 03. Method of administration of a gene of interest to a vascular tissue. Duverger, Nicolas, et al. 604/522; 435/320.1 604/509 A61M031/00.
4. 20030096747. 23 May 02. 22 May 03. Methods and compositions for preventing and treating male erectile dysfunction and female sexual arousal disorder. Lue, Tom F., et al. 514/12; 424/93.2 514/44 A61K048/00 A61K038/18.
5. <u>20030012768</u> . 11 Jul 01. 16 Jan 03. Connective tissue growth factor-2. Li, Haodong, et al. 424/93.2; 435/456 514/44 A61K048/00 C12N015/861.
☐ 6. <u>20020187132</u> . 30 Apr 01. 12 Dec 02. Cardiac gene transfer. Mcgregor, Christopher G.A., et al. 424/93.21; 435/455 A61K048/00 C12N015/85.
7. 20020176847. 08 Mar 02. 28 Nov 02. Methods for inhibiting macrophage colony stimulating factor and c-FMS-dependent cell signaling. Rajavashisth, Tripathi. 424/93.2; 435/456 514/44 A61K048/00 C12N015/86.
8. 20020160951. 19 Jul 01. 31 Oct 02. Methods and compositions for preventing and treating male erectile dysfunction and female sexual arousal disorder. Lue, Tom F., et al. 514/12; 514/44 A61K048/00 A61K038/18.
9. <u>20020155101</u> . 06 Sep 01. 24 Oct 02. Cardiac arrhythmia treatment methods. Donahue, J. Kevin, et al. 424/93.21; 435/6 514/44 C12Q001/68 A61K048/00.
☐ 10. <u>20020131959</u> . 14 Mar 01. 19 Sep 02. Means and methods for the modulation of arteriogenesis. Buschmann, Ivo, et al. 424/93.21; 424/85.1 424/85.2 514/2 514/44 A61K048/00 A61K038/18 A61K038/19 A61K038/20.
☐ 11. <u>RE37933</u> . 21 Dec 00; 10 Dec 02. Viral vectors and their use for treating hyperproliferative disorders, in particular restenosis. Branellec; Didier, et al. 424/93.2; 435/320.1 435/325 435/375 435/456 514/44 536/23.5. A61K035/76 A61K048/00 C12N015/86 C12N015/63.
☐ 12. <u>6271211</u> . 21 Mar 00; 07 Aug 01. Gene therapy for regulating penile smooth muscle tone. Christ; George J., et al. 514/44; 435/320.1 435/325 435/455 530/350 536/23.1 536/23.5. A01N043/04 A61K031/70 C12N015/00 C12N015/09 C12N015/63.
13. 6239117 . 21 Mar 00; 29 May 01. Gene therapy for regulating bladder smooth muscle tone



Christ; George J., et al. 514/44; 435/320.1 435/325 435/455 530/350 536/23.1 536/23.5 800/8. A01N043/04 A61K031/70.

- ☑ 14. <u>6174871</u>. 10 Aug 98; 16 Jan 01. Gene therapies for enhancing cardiac function. Hammond; H. Kirk, et al. 514/44; 424/93.6 435/320.1 536/23.5. A01N043/04 A01N063/00 A61K031/70 C12N015/00 C12N015/09 C12N015/63 C12N015/70 C12N015/74.
- ☑ 15. <u>5858990</u>. 04 Mar 97; 12 Jan 99. Fas ligand compositions for treatment of proliferative disorders. Walsh; Kenneth. 514/44; 435/320.1 435/375 435/377 435/6 435/69.1. A61K048/00 C12N015/11.
- ☐ 16. <u>5851521</u>. 30 Sep 96; 22 Dec 98. Viral vectors and their use for treating hyperproliferative disorders, in particular restenosis. Branellec; Didier, et al. 424/93.2; 435/320.1 435/325 435/375 435/456 514/44 536/23.5. A61K035/76 A61K048/00 C12N015/86 C12N015/63.

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15 and L6	16

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### (FILE 'HOME' ENTERED AT 13:52:11 ON 19 AUG 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 13:52:41 ON 19 AUG 2003 L19353 S ADENO-ASSOCIATED (W) VIRUS OR AAV 624387 S (CARDIOVASCULAR OR CARDIAC OR HEART) (3A) (DISEASE OR DEFECT OR L2L3 187 S L1 AND L2 94404 S GENE (W) THERAPY L4L5 4220 S L1 AND L4 295912 S (CORONARY OR SINUS) (3A) ARTERY L6 39 S L5 AND L6 L7L833 DUP REM L7 (6 DUPLICATES REMOVED) L9 19 S L3 AND L8 L10 19 DUP REM L9 (0 DUPLICATES REMOVED) L1113176 S INFUS? (6A) ARTERY L12 5 S L3 AND L11 L13 3 DUP REM L12 (2 DUPLICATES REMOVED) => d au ti so ab 1-19 l9 ANSWER 1 OF 19 L9 MEDLINE on STN ΑU Asfour B; Baba H A; Scheld H H; Hruban R H; Hammel D; Byrne B J Uniform long-term gene expression using adeno-associated virus (AAV) by ex vivo recirculation in rat-cardiac isografts. THORACIC AND CARDIOVASCULAR SURGEON, (2002 Dec) 50 (6) 347-50.

Journal code: 7903387. ISSN: 0171-6425. AB

BACKGROUND: Gene therapy in cardiovascular disease promises to be of great impact. The ideal vector for the therapeutic gene transfection remains to be determined. The aim of the present study was to investigate the efficacy of gene transfer using adeno-associated virus vectors carrying the lacZ-reporter gene (AAV-lacZ) in a previously described coronary recirculation model. METHODS: Beating Lewis rat hearts perfused with oxygenated Krebs-Henseleit solution were harvested, after which an atrial septal defect (ASD) was created. All vessels were tied, and AAV -lacZ was injected into the aortic root. The solution was recirculated through the ASD to the left side of the heart and pumped back to the coronary arteries by the left ventricle. Incubation was allowed for 20 min at 15 degrees C, and the hearts were subsequently transplanted heterotopically in syngeneic rats. Three increasing doses (109, 1,010, 1,011 e. u.) of AAV-lacZ virus vectors were used to study the rate of gene transfer. All hearts were harvested after 7-60 days and evaluated histologically for expression of the lacZ-gene. RESULTS: Dose-dependent gene transfer was observed. Even after 60 days, there was no obvious decline in gene expression. CONCLUSION: Adeno-associated virus vectors offer effective and uniform gene transfer in the myocardium after transcoronary injection and recirculation. Due to the lack of immune response previously described, no decrease in gene expression can be observed up to 60 days after injection.

- L9 ANSWER 2 OF 19 MEDLINE on STN
- ΑU Kaplitt M G; Xiao X; Samulski R J; Li J; Ojamaa K; Klein I L; Makimura H; Kaplitt M J; Strumpf R K; Diethrich E B
- ΤI Long-term gene transfer in porcine myocardium after coronary infusion of an adeno-associated virus vector.
- ANNALS OF THORACIC SURGERY, (1996 Dec) 62 (6) 1669-76. SO Journal code: 15030100R. ISSN: 0003-4975.
- BACKGROUND: Viral vector-mediated gene transfer into the heart represents AΒ a potentially powerful tool for studying both cardiac physiology as well

as gene therapy of cardiac disease

We report here the use of a defective viral vector, which expresses no viral gene products, for gene transfer into the mammalian heart. Previous studies have used recombinant viral vectors, which retained viral genes and yielded mostly short-term expression, often with significant inflammation. METHODS: An adeno-associated virus vector was used that contains no viral genes and is completely free of contaminating helper viruses. The adenoassociated virus vector was applied to rat hearts by direct intramuscular injection; adeno-associated virus was also infused into pig hearts in vivo via percutaneous intraarterial infusion into the coronary vasculature using routine catheterization techniques. RESULTS: Gene transfer into rat heart yielded no apparent inflammation, and expression was observed for at least 2 months after injection. Infusion into pig circumflex coronary arteries resulted in successful transfer and expression of the reporter gene in cardiac myocytes without apparent toxicity or inflammation; gene expression was observed for at least 6 months after infusion. CONCLUSIONS: We report the use of adenoassociated virus vectors in the cardiovascular system as well as successful myocardial gene transfer after percutaneous coronary artery infusion of viral vectors in a large, clinically relevant mammalian model. These results suggest that safe and stable gene transfer can be achieved in the heart using standard outpatient cardiac catheterization techniques.

- ANSWER 3 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN L9
- IN Dzau, Victor; Melo, Luis G.; Perrella, Mark A.; Agrawal, Reitu
- ΤI Methods of treating cardiac disorders
- PCT Int. Appl., 58 pp. so CODEN: PIXXD2
- The invention features methods and compns. for treating ischemic and AB reperfusion related injury such as cardiac disorders. Cardiomyocyte cell death is prevented by administering a compn. contg. a nucleic acid encoding a human heme oxygenase-1 polypeptide or extracellular superoxide dismutase polypeptide or biol. active fragment thereof. Among the examples provided are: myocardial tissue from oxidative stress by gene HO-1 delivery via an adeno-assocd. vector, effect of HO-1 gene transfer on oxidative stress-induced lipid peroxidn. and expression of apoptosis-related proteins and inflammatory cytokines, and myocardial protective action of extracellular superoxide dismutase gene transfer.
- L9 ANSWER 4 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Hammond, H. Kirk
- TI Techniques and compositions for treating cardiovascular disease by in vivo angiogenic polypeptide-encoding gene delivery
- PCT Int. Appl., 129 pp. CODEN: PIXXD2
- Methods are provided for treating patients with cardiovascular disease, including heart disease and peripheral vascular disease. The preferred methods of the invention involve in vivo delivery of genes encoding angiogenic proteins or peptides to the myocardium or to peripheral ischemic tissue, by introduction of a vector contg. the gene into a blood vessel supplying the heart or into a peripheral ischemic tissue.
- ANSWER 5 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN L9
- IN Wu, Kenneth K.
- TI Vectors, compositions and methods for treating a vascular disorder PCT Int. Appl., 72 pp.  $\,$
- SO CODEN: PIXXD2
- The present invention discloses vectors comprising a cyclooxygenase sequence, a prostaglandin synthase sequence, or both. The invention

further discloses methods of making such vectors, and compns. comprising such vectors. Methods for treating a patient afflicted with a vascular disorder by use of said vectors and compns. are also disclosed.

- L9 ANSWER 6 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Hammond, H. Krik; Insel, Paul A.; Ping, Peipei; Post, Steven R.; Gao, Meihua
- TI Gene therapy for congestive heart failure
- SO U.S. Pat. Appl. Publ., 69 pp., Cont.-in-part of U.S. Ser. No. 472,667. CODEN: USXXCO
- AB The present invention relates to methods and compns. for enhancing cardiac function in mammalian hearts by inserting transgenes that increase beta-adrenergic responsiveness within the myocardium. The present invention can thus be used in the treatment of heart disease, esp. congestive heart failure.
- L9 ANSWER 7 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Gao, Mei Hua
- TI Dual recombinant **gene therapy** compositions and methods of use
- SO PCT Int. Appl., 74 pp. CODEN: PIXXD2
- AB The present invention relates to novel compns. and methods for the treatment of cardiovascular disease. More particularly, the invention relates to gene therapy compns. comprising at least two transgenes encoding angiogenic proteins or peptides. In one aspect the two transgenes are provided in a single gene delivery vector. Alternatively, the compn. comprises at least two vectors, each vector comprising a transgene encoding a different angiogenic protein or peptide. The invention also relates to methods of treating cardiovascular disease using the gene therapy compns.; kits for gene delivery; and pharmaceutical compns.
- L9 ANSWER 8 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Hsu, Yen-Ming; Garber, Ellen
- TI CD154 variants
- SO PCT Int. Appl., 41 pp. CODEN: PIXXD2
- AB Methods of decreasing (e.g., inhibiting) the expression of wild-type CD154 on the surface of a target cell and methods of treating a patient suffering from or predisposed to a CD154-mediated disease. In these methods, a nucleic acid construct that directs expression of a mutant CD154 lacking at least a portion of the tumor necrosis factor homologous domain ("TNFH") is introduced into a target cell (such as a T helper cell or a cytotoxic T cell). The expressed mutant CD154 binds to wild-type CD154 inside the cell, rendering the wild-type protein unable to reach the cell surface.
- L9 ANSWER 9 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Guo, Kun; Pagnoni, Marco F.; Clark, Kenneth L.; Ivashchenko, Yuri D.
- TI Human protein kinase Akt3 and cDNAs encoding it and the use of the enzyme in treatment of hypoxia, apoptosis or necrosis
- SO PCT Int. Appl., 73 pp. CODEN: PIXXD2
- AB The present invention relates to human Akt3 proteins and polypeptides. The invention also relates to isolated nucleic acids encoding human Akt3, to vectors contg. them and to their therapeutic uses, in particular for gene therapy. Expression of Akt3 inhibits cell death assocd. with hypoxia, apoptosis or necrosis.
- L9 ANSWER 10 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Yen, Frances; Erickson, Mary Ruth; Fruebis, Joachim; Bihain, Bernard
- TI Methods of screening for compounds that modulate the LSR (lipolysis

stimulated receptor)-leptin interaction and their use in the prevention and treatment of obesity-related diseases

SO PCT Int. Appl., 247 pp. CODEN: PIXXD2

- The present invention is drawn to methods of screening for new compds. for AB the treatment of obesity and obesity-related diseases and disorders, as well as methods of treating obesity-related diseases and disorders, based on the discovery of the role of the leptin-LSR interaction in obesity. The lipolysis stimulated receptor (LSR) displays a high affinity for unmodified triglyceride-rich lipoproteins and is involved in the partitioning of dietary lipids among the liver, adipose tissue and muscle. Leptin and the leptin fragment described herein were found to diminish the postprandial lipemic response in dbPas/dbPa5 mice which lack the leptin OB receptor, thereby showing that leptin signaling can be independent of the OB receptor. Leptin increases the activity of LSR, binds directly to LSR, and that leptin binding leads to leptin degrdn. LSR is actually at least two receptors, one for triglyceride-rich lipoproteins, and one for leptin. The three subunits that make up LSR, .alpha., .beta., and .alpha.', actually combine in at least two ways: (1) .alpha. and .beta. together make up the LSR receptor for triglyceride-rich lipoproteins, and (2) .alpha.' is a necessary part of the LSR receptor for leptin, that may include .beta. as well. Thus, it is now clear that assays can be designed for identifying modulators or receptors/binding partners/signalling cascade members that are specific for the triglyceride-related activity of LSR or for the leptin-related activity of LSR or both. Further, the invention features the discovery of a 22 amino acid region of human leptin that modulates LSR activity in vitro and in vivo in the same way as the intact human leptin, thus allowing the use of only this crit. region in assays for modulators of the leptin-LSR interaction, and new leptin receptors and binding partners. The new leptin fragment can also be used in disease treatment since it is active in mice at a physiol.-relevant level.
- L9 ANSWER 11 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN
- AU Su, Hua; Lu, Ronghua; Kan, Yuet Wai
- TI Adeno-associated viral vector-mediated vascular endothelial growth factor gene transfer induces neovascular formation in ischemic heart
- Proceedings of the National Academy of Sciences of the United States of America (2000), 97(25), 13801-13806 CODEN: PNASA6; ISSN: 0027-8424
- AB Vascular endothelial growth factor (VEGF) plays important roles in physiol. and pathol. angiogenesis. Recent studies have demonstrated that direct injection of VEGF protein, plasmid DNA, or an adenoviral vector encoding the VEGF gene into ischemic myocardium or limb can induce collateral blood vessel formation and improve perfusion of the ischemic areas. However, these approaches have limitations ranging from a short-lasting effect to angioma formation. In this study, we investigated the feasibility of using adeno-assocd. viral (AAV) vectors to deliver VEGF genes to mouse myocardium. A cytomegalovirus promoter was used to drive genes for a human VEGF isoform, VEGF165, and Lacz. A mouse myocardial ischemic model was generated by ligation of the anterior descending coronary artery. Approx. 1011 copies of the AAV-VEGF vector mixed with 1010 copies of AAV-LacZ were injected to one site of normal myocardium and a total of 1011 copies of AAV-VEGF were injected to multiple sites of myocardium around the ischemic region. LacZ gene expression was obsd. up to 3 mo after the vector inoculation. After AAV-VEGF inoculation, neoangiogenesis was obsd. in the ischemic heart model but not in normal heart tissue. inflammatory-cell infiltration was not obsd. in the AAV-VEGFand AAV-LacZ-inoculated hearts, and angioma-like structure was not obsd. These results indicated that injection of the AAV vector directly to myocardium could mediate efficient gene transfer and transgene expression and that VEGF gene delivered by AAV vector can induce angiogenesis in ischemic myocardium.

- L9 ANSWER 12 OF 19 CAPLUS .COPYRIGHT 2003 ACS on STN
- IN Guo, Kun; Pagnoni, Marco F.; Clark, Kenneth L.; Ivashchenko, Yuri D.
- TI Human protein kinase Akt3 nucleic acids, polypeptides, and biological functions and applications
- SO PCT Int. Appl., 73 pp. CODEN: PIXXD2
- The present invention relates to human Akt3 proteins and polypeptides. The 3rd isoform of AH/PH domain-contg. serine/threonine kinase (Akt3) was cloned from a human cDNA library and shown to comprise a 465-amino acid protein that is ubiquitously expressed with the highest level of expression obsd. in the heart. Akt3 protein possesses Akt activity and inhibits apoptotic stimulating kinase 1 (ASK1)-induced cell death in HEK293 cells. The invention also relates to isolated nucleic acids encoding human Akt3, to vectors contg. them and to their therapeutic uses, in particular for gene therapy. Expression of Akt3 inhibits cell death assocd. with hypoxia, apoptosis or necrosis.
- L9 ANSWER 13 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Leiden, Jeffrey M.; Svensson, Eric
- TI Efficient and stable in vivo gene transfer to cardiomyocytes using recombinant adeno-associated virus vectors
- SO PCT Int. Appl., 20 pp. CODEN: PIXXD2
- AB Recombinant adeno-assocd. virus (rAAV)
  vectors are used to transduce cardiomyocytes in vivo by infusing the rAAV
  into a coronary artery or coronary
  sinus. RAAV infection is not assocd. with detectable myocardial
  inflammation or myocyte necrosis. Thus, rAAV is a useful vector for the
  stable expression of therapeutic genes in the myocardium and can be used
  to deliver genes for inducing angiogenesis, inhibiting angiogenesis,
  stimulating cell proliferation, inhibiting cell proliferation and/or
  treating or ameliorating other cardiovascular conditions.
- L9 ANSWER 14 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Hammond, H. Kirk
- TI Gene transfer-mediated angiogenesis therapy and techniques for intravascular gene delivery
- SO PCT Int. Appl., 46 pp. CODEN: PIXXD2
- AB Transgene-inserted vectors are effectively used for in vivo gene therapy for peripheral vascular disease, heart disease and other conditions, by direct injection of the vector into arteries supplying the tissue to be targeted, preferably in combination with a vasoactive agent that is infused into the artery prior to or coincident with delivery of the vector.
- L9 ANSWER 15 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Woolf, Tod M.
- TI Novel methods of stabilizing mRNA
- SO PCT Int. Appl., 53 pp. CODEN: PIXXD2
- The invention describes novel methods of altering eukaryotic mRNA, resulting in its stabilization against nucleases and enabling it to transiently express proteins of interest in a cell. Stabilization of the mRNA of the invention can be achieved by end blocking modifications, sequence modifications, and/or chem. modifications. In one aspect, the invention pertains to modified mRNA mols. encoding therapeutically relevant proteins and the possible use of said mRNA in sense RNA therapy.
- L9 ANSWER 16 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Hammond, H. Kirk; Kelly, Tamsin L.
- TI Techniques and compositions for treating heart failure and ventricular remodeling by in vivo delivery of angiogenic transgenes

- SO PCT Int. Appl., 87 pp. CODEN: PIXXD2
- AB Methods are provided for treating patients with congestive heart failure (including dilated cardiomyopathy and congestive heart failure assocd. with severe coronary artery disease), and for preventing or alleviating deleterious ventricular remodeling after myocardial infarction. The preferred methods of the present invention involve in vivo delivery of genes encoding angiogenic proteins or peptides to the myocardium by direct injection of a vector contg. the gene into a blood vessel supplying the heart. Preferred angiogenic factors include members of the fibroblast growth factor family, the vascular endothelial growth factor family, the platelet-derived growth factor family, and the insulin-like growth factor family. Thus, a helper-independent replication-defective human adenovirus 5 system is used effectively to transfect a large percentage of myocardial cells in vivo by a single intracoronary injection. Such a delivery technique is used to effectively target vectors to the myocardium of a large mammal heart, using the myosin light chain 2 or myosin heavy chain promoters specific for cardiac myocytes. Transient adenovirus-mediated gene transfer is therapeutically adequate for treating cardiovascular conditions. Within 14 days after gene transfer of fibroblast growth factor 5 (FGF5) into the myocardium, blood flow to the ischemic bed had increased 2-fold and the effect persisted for .gtoreq.12 wk. Wall thickening also increased within 2 wks after gene transfer and persisted for .gtoreq.12 wk.
- L9 ANSWER 17 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Hammond, H. Kirk; Insel, Paul A.; Ping, Peipei; Post, Steven R.; Gao, Meihua
- TI Gene therapy for congestive heart failure using genes for .beta.-adrenoceptors to increase responsiveness to .beta.-adrenergic agonists
- SO PCT Int. Appl., 114 pp. CODEN: PIXXD2
- AB Cardiac function is improved in the treatment of congestive heart failure by introduction of a gene that increase .beta.-adrenergic responsiveness within the myocardium. The gene may be any of several involved in the .beta.-adrenoceptor signal transduction chain and may include the receptor itself, a G protein receptor kinase inhibitor, or an adenyl cyclase. The preferred gene is one for a cardiac isoenzyme of adenylate cyclase. Studies in a pig model of congestive heart demonstrated a role for adenyl cyclase in cardiac function and in congestive heart failure. A .beta.-adrenoceptor and G-protein receptor kinase were also shown to be involved in this model: A method for rapid screening of constructs for efficiency of gene transfer using rat ventricular myocytes is described. Succesful transfer of genes from adenoviral vectors into the myocardium is demonstrated in a swine heart failure model.
- L9 ANSWER 18 OF 19 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN AU Su, Hua; Arakawa-Hoyt, Janice; Kan, Yuet Wai (1)
- TI Adeno-associated viral vector-mediated hypoxia response element-regulated gene expression in mouse ischemic heart model.
- Proceedings of the National Academy of Sciences of the United States of America, (July 9, 2002) Vol. 99, No. 14, pp. 9480-9485. http://www.pnas.org. print. ISSN: 0027-8424.
- AB Intramyocardial injection of genes encoding angiogenic factors could provide a useful approach for the treatment of ischemic heart disease. However, uncontrolled expression of angiogenic factors in vivo may cause some unwanted side effects, such as hemangioma formation, retinopathy, and arthritis. It may also induce occult tumor growth and artherosclerotic plaque progression. Because hypoxia-inducible factor 1 is up-regulated in a variety of hypoxic conditions and it regulates gene expression by binding to a cis-acting hypoxia-responsive element (HRE), we propose to use HRE, found in the 3' end of the erythropoietin gene to

control gene expression in ischemic myocardium. A concatemer of nine copies of the consensus sequence of HRE isolated from the erythropoietin enhancer was used to mediate hypoxia induction. We constructed two adeno-associated viral vectors in which LacZ and vascular endothelial growth factor (VEGF) expressions were controlled by this HRE concatemer and a minimal simian virus 40 promoter. Both LacZ and VEGF expression were induced by hypoxia and/or anoxia in several cell lines transduced with these vectors. The functions of these vectors in ischemic myocardium were tested by injecting them into normal and ischemic mouse myocardium created by occlusion of the left anterior descending coronary artery. The expression of LacZ gene was induced eight times and of VEGF 20 times in ischemic myocardium compared with normal myocardium after the viral vector transduction. Hence, HRE is a good candidate for the control of angiogenic factor gene expression in ischemic myocardium.

ANSWER 19 OF 19 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN L9 ΑU Melo, Luis Gabriel (1); Agrawal, Reitu A. (1); Zhang, Lunan (1); Rezvani, Mojgan (1); Mangi, Abeel A. (1); Dell'Acqua, Giorgio (1); Yet, Shaw-Fang (1); Perrella, Mark A.; Dzau, Victor J.

ΤI Intramyocardial delivery of heme oxygenase-1 gene by adenoassociated virus provides long-term protection from ischemia/reperfusion injury.

SO Circulation, (October 23, 2001) Vol. 104, No. 17 Supplement, pp. II.35. http://circ.ahajournals.org/. print. Meeting Info.: Scientific Sessions 2001 of the American Heart Association Anaheim, California, USA November 11-14, 2001 ISSN: 0009-7322.

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    ANSWER 3 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN
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AN 2002:946119 CAPLUS

DN 138:19497

ΤI Methods of treating cardiac disorders

IN Dzau, Victor; Melo, Luis G.; Perrella, Mark A.; Agrawal, Reitu

PΑ The Brigham and Women's Hospital, Inc., USA

SO PCT Int. Appl., 58 pp. CODEN: PIXXD2

DT Patent

LA English

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L9 ANSWER 4 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN

AN 2002:868781 CAPLUS

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ΤI Techniques and compositions for treating cardiovascular

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disease by in vivo angiogenic polypeptide-encoding gene delivery
     Hammond, H. Kirk
IN
     The Regents of the University of California, USA
PΑ
     PCT Int. Appl., 129 pp.
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     CODEN: PIXXD2
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AN
     2002:716105 CAPLUS
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ΤI
     Vectors, compositions and methods for treating a vascular disorder
IN
     Wu, Kenneth K.
     Board of Regents, the University of Texas System, USA
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     PCT Int. Appl., 72 pp.
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     Gene therapy for congestive heart failure
     Hammond, H. Krik; Insel, Paul A.; Ping, Peipei; Post, Steven R.; Gao,
IN
     Meihua
PA
     U.S. Pat. Appl. Publ., 69 pp., Cont.-in-part of U.S. Ser. No. 472,667.
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     Dual recombinant gene therapy compositions and methods
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     of use
IN
     Gao, Mei Hua
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     Collateral Therapeutics, Inc., USA
     PCT Int. Appl., 74 pp.
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     CD154 variants
IN
     Hsu, Yen-Ming; Garber, Ellen
     Biogen, Inc., USA
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     PCT Int. Appl., 41 pp.
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     Human protein kinase Akt3 and cDNAs encoding it and the use of the enzyme
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     in treatment of hypoxia, apoptosis or necrosis
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     Guo, Kun; Pagnoni, Marco F.; Clark, Kenneth L.; Ivashchenko, Yuri D.
     Aventis Pharmaceuticals Products Inc., USA
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      2000:688350 CAPLUS
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      Human protein kinase Akt3 nucleic acids, polypeptides, and biological
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      Guo, Kun; Pagnoni, Marco F.; Clark, Kenneth L.; Ivashchenko, Yuri D.
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      2000:456818 CAPLUS
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ΤI
      Efficient and stable in vivo gene transfer to cardiomyocytes using
      recombinant adeno-associated virus vectors
IN
      Leiden, Jeffrey M.; Svensson, Eric
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      Arch Development Corp., USA
      PCT Int. Appl., 20 pp.
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     Gene transfer-mediated angiogenesis therapy and techniques for
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IN
     Hammond, H. Kirk
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     The Regents of the University of California, USA
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     Novel methods of stabilizing mRNA
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     Woolf, Tod M.
     Sequitur, Inc., USA
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     ANSWER 16 OF 19 CAPLUS COPYRIGHT 2003 ACS on STN
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     1998:744977 CAPLUS
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ΤI
     Techniques and compositions for treating heart failure and ventricular
     remodeling by in vivo delivery of angiogenic transgenes
IN
     Hammond, H. Kirk; Kelly, Tamsin L.
PΑ
     The Regents of the University of California, USA
SO
     PCT Int. Appl., 87 pp.
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ΤI
      Gene therapy for congestive heart failure using genes
      for .beta.-adrenoceptors to increase responsiveness to .beta.-adrenergic
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IN
      Hammond, H. Kirk; Insel, Paul A.; Ping, Peipei; Post, Steven R.; Gao,
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PA
      Regents of the University of California, USA; Collateral Therapeutics;
      Hammond, H. Kirk; Insel, Paul A.; Ping, Peipei; Post, Steven R.; Gao,
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 AN
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 TI
      Efficient and stable in vivo gene transfer to cardiomyocytes using
      recombinant adeno-associated virus vectors
 IN
      Leiden, Jeffrey M.; Svensson, Eric
 PA
      Arch Development Corp., USA
      PCT Int. Appl., 20 pp.
 SO
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      Recombinant adeno-assocd. virus (rAAV)
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      vectors are used to transduce cardiomyocytes in vivo by infusing
      the rAAV into a coronary artery or coronary sinus. RAAV
      infection is not assocd. with detectable myocardial inflammation or
      myocyte necrosis. Thus, rAAV is a useful vector for the stable expression
      of therapeutic genes in the myocardium and can be used to deliver genes
      for inducing angiogenesis, inhibiting angiogenesis, stimulating cell
      proliferation, inhibiting cell proliferation and/or treating or
      ameliorating other cardiovascular conditions.
 RE.CNT 4
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      Gene transfer-mediated angiogenesis therapy and techniques for
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 IN
      Hammond, H. Kirk
 PA
      The Regents of the University of California, USA
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      PCT Int. Appl., 46 pp.
      CODEN: PIXXD2
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AB
     Transgene-inserted vectors are effectively used for in vivo gene therapy
     for peripheral vascular disease, heart disease
     and other conditions, by direct injection of the vector into arteries
     supplying the tissue to be targeted, preferably in combination with a
     vasoactive agent that is infused into the artery prior
     to or coincident with delivery of the vector.
L13
     ANSWER 3 OF 3
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     97116282
                   MEDLINE
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     97116282
                 PubMed ID: 8957370
TΙ
     Long-term gene transfer in porcine myocardium after coronary infusion of
     an adeno-associated virus vector.
ΑU
     Kaplitt M G; Xiao X; Samulski R J; Li J; Ojamaa K; Klein I L; Makimura H;
     Kaplitt M J; Strumpf R K; Diethrich E B
CS
     Department of Surgery, New York Hospital-Cornell University Medical
     College, New York, USA.
so
     ANNALS OF THORACIC SURGERY, (1996 Dec) 62 (6) 1669-76.
     Journal code: 15030100R. ISSN: 0003-4975.
CY
     United States
DТ
     Journal; Article; (JOURNAL ARTICLE)
T.A
     English
FS
     Abridged Index Medicus Journals; Priority Journals
EΜ
     199701
ED
     Entered STN: 19970128
     Last Updated on STN: 19970128
     Entered Medline: 19970109
AB
     BACKGROUND: Viral vector-mediated gene transfer into the heart represents
     a potentially powerful tool for studying both cardiac physiology as well
     as gene therapy of cardiac disease. We report here
     the use of a defective viral vector, which expresses no viral gene
     products, for gene transfer into the mammalian heart. Previous studies
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have used recombinant viral vectors, which retained viral genes and

yielded mostly short-term expression, often with significant inflammation. METHODS: An adeno-associated virus vector was used that contains no viral genes and is completely free of contaminating helper viruses. The adeno-associated virus vector was applied to rat hearts by direct intramuscular injection; adeno-associated virus was also infused into pig hearts in vivo via percutaneous intraarterial infusion into the coronary vasculature using routine catheterization techniques. RESULTS: Gene transfer into rat heart yielded no apparent inflammation, and expression was observed for at least 2 months after injection. Infusion into pig circumflex coronary arteries resulted in successful transfer and expression of the reporter gene in cardiac myocytes without apparent toxicity or inflammation; gene expression was observed for at least 6 months after infusion. CONCLUSIONS: We report the use of adeno-associated virus vectors in the cardiovascular system as well as successful myocardial gene transfer after percutaneous coronary artery infusion of viral vectors in a large, clinically relevant mammalian model. These results suggest that safe and stable gene transfer can be achieved in the heart using standard outpatient cardiac catheterization techniques.

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  ISSN: 0009-7322.
- L8 ANSWER 2 OF 33 CAPLUS COPYRIGHT 2003 ACS on STN
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- SO Laboratory Investigation (2003), 83(8), 1097-1104 CODEN: LAINAW; ISSN: 0023-6837
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- L8 ANSWER 4 OF 33 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Hammond, H. Kirk
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- L8 ANSWER 8 OF 33 CAPLUS COPYRIGHT 2003 ACS on STN
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- TI . Gene therapy for congestive heart failure
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- SO Proceedings of the National Academy of Sciences of the United States of America, (July 9, 2002) Vol. 99, No. 14, pp. 9480-9485. http://www.pnas.org.print. ISSN: 0027-8424.
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- TI Gene therapy strategy for long-term myocardial protection using adeno-associated virus -mediated delivery of heme oxygenase gene.
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- SO THORACIC AND CARDIOVASCULAR SURGEON, (2002 Dec) 50 (6) 347-50. Journal code: 7903387. ISSN: 0171-6425.
- L8 ANSWER 12 OF 33 CAPLUS COPYRIGHT 2003 ACS on STN
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- TI CD154 variants
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- L8 ANSWER 13 OF 33 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Guo, Kun; Pagnoni, Marco F.; Clark, Kenneth L.; Ivashchenko, Yuri D.
- TI Human protein kinase Akt3 and cDNAs encoding it and the use of the enzyme in treatment of hypoxia, apoptosis or necrosis
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- L8 ANSWER 14 OF 33 CAPLUS COPYRIGHT 2003 ACS on STN
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- TI Recombinant adeno-associated virus (rAAV) expressing proteins involved in preventing cell proliferation, thrombosis, and/or cell migration in a vascular graft
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- TI Inhibition of vascular smooth cell proliferation with transfer of wild-type p53 gene using vector based on adeno-associated virus plasmid
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- TI Localized delivery of adeno-associated virus vector expressing human extracellular superoxide dismutase gene confers long term protection against ischemia-reperfusion injury to the rat heart.
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  ISSN: 0009-7322.
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  ISSN: 0009-7322.
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- IN Guo, Kun; Pagnoni, Marco F.; Clark, Kenneth L.; Ivashchenko, Yuri D.
- TI Human protein kinase Akt3 nucleic acids, polypeptides, and biological functions and applications
- SO PCT Int. Appl., 73 pp. CODEN: PIXXD2
- L8 ANSWER 22 OF 33 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Leiden, Jeffrey M.; Svensson, Eric
- TI Efficient and stable in vivo gene transfer to cardiomyocytes using recombinant adeno-associated virus vectors
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- L8 ANSWER 23 OF 33 CAPLUS COPYRIGHT 2003 ACS on STN
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- IN Hammond, H. Kirk

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- SO PCT Int. Appl., 46 pp. CODEN: PIXXD2
- L8 ANSWER 25 OF 33 CAPLUS COPYRIGHT 2003 ACS on STN
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- L8 ANSWER 30 OF 33 CAPLUS COPYRIGHT 2003 ACS on STN
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- SO PCT Int. Appl., 114 pp. CODEN: PIXXD2
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- IN Goldenberg, Tsvi; Tritz, Richard
- TI Ribozyme therapy for the inhibition of restenosis
- SO U.S., 20 pp., Cont.-in-part of U.S. Ser. No. 207,649, abandoned. CODEN: USXXAM
- L8 ANSWER 32 OF 33 CAPLUS COPYRIGHT 2003 ACS on STN
- IN Goldenberg, Tsvi; Tritz, Richard
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ΑU
     Kaplitt M G; Xiao X; Samulski R J; Li J; Ojamaa K; Klein I L; Makimura H;
     Kaplitt M J; Strumpf R K; Diethrich E B
ΤI
     Long-term gene transfer in porcine myocardium after coronary infusion of
     an adeno-associated virus vector.
     ANNALS OF THORACIC SURGERY, (1996 Dec) 62 (6) 1669-76.
so
     Journal code: 15030100R. ISSN: 0003-4975.
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     130:22238
     Enzymic ribozyme treatment of diseases or cancers related to expression of
TΙ
     c-raf gene
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